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Inflammatory cytokines IL-1β and TNF-α regulate p75^{NTR} expression in CNS neurons and astrocytes by distinct cell-type-specific signalling mechanisms

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SUPPLEMENTARY ONLINE DATA

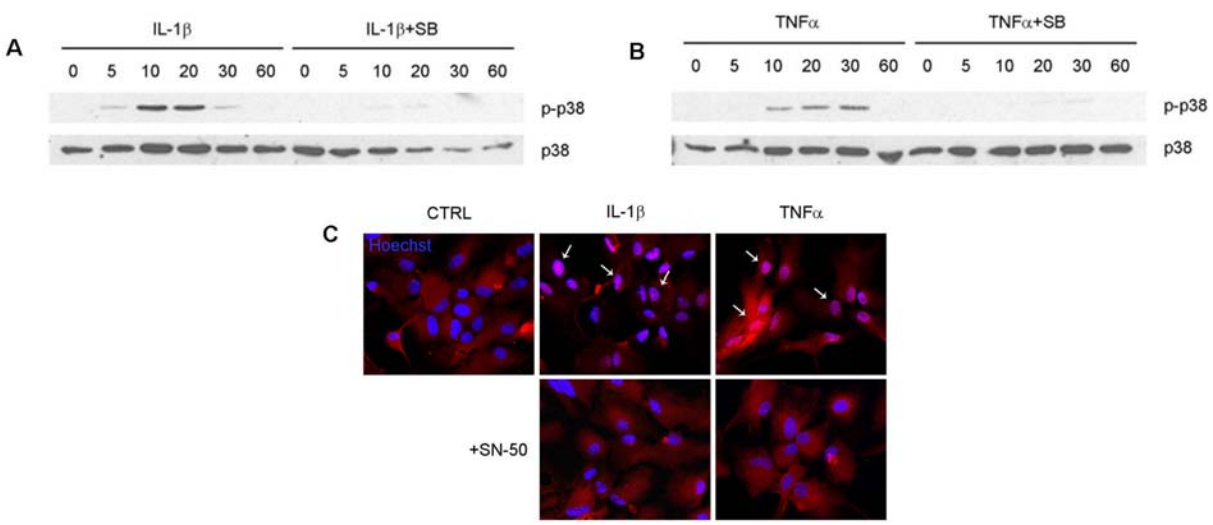

Figure S1 **Confirmation that inhibitors of p38 MAPK and NF-κB inhibit their respective pathways**
Treatment of hippocampal astrocytes with **(A)** IL-1β or **(B)** TNF-α in the absence or presence of the p38 MAPK inhibitor showed that SB203580 inhibited p38 MAPK phosphorylation induced by either ligand. **(C)** To confirm the inhibitory effect of SN-50 on NF-κB nuclear translocation, astrocytes were pretreated with vehicle or with 10 μM SN-50 for 30 min and then treated with IL-1β or TNF-α for 30 min. Cells were fixed and stained for p65, and nuclei were labelled with Hoechst stain. Nuclear staining of p65 was observed in IL-1β- or TNF-α-treated astrocytes without SN-50 (indicated by arrows; note the pink colour of the nuclei), and nuclear translocation of p65 was blocked by SN-50 (bottom panels).

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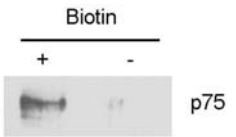

Figure S2 Hippocampal neurons were treated with or without biotin
Cells were processed as indicated in Figure 6 of the main paper, lysates were precipitated with streptavidin and analysed by Western blot for $p75^{NTR}$ levels. In the absence of biotin, no $p75^{NTR}$ was detected.

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